INTERNATIONAL JOURNAL OF ADVANCED BIOLOGICAL RESEARCH

www.scienceandnature.org

HIGH SENSITIVITY C – REACTIVE PROTEIN AND BODY MASS INDEX AS PREDICTORS OF OBESITY IN POSTMENOPAUSAL IRAQI WOMEN

Noor Abdul Rahman Karim, Jabbar H. Yenzeel & Sabah N. Al-Wachi
Department of Biology, College of Sciences, University of Baghdad, Baghdad, Iraq
*Corresponding author email: dr.jabbaryenzeel@gmail.com

ABSTRACT
Following postmenopausal women obesity there are changes in values of different parameters like triglyceride, high sensitive C-reactive protein (hs-CRP) and anthropometric measurement (body mass index (BMI), waist hip ratio (WHR), waist height ratio (WHtR) and neck circumference (NC)). So, this study was designed to observe the pattern of parameters in postmenopausal obesity in Iraqi women. Fifty female patients suffering from obesity disease with mean age (54.68 ± 0.89) were taken during attended the consulting clinic in: Al- Kindy Teaching Hospital and Obesity Research and Treatment unit Al-Kindy College of Medicine/University of Baghdad. According to level of hs-CRP the women were divided into: (17 women with hs-CRP> 6mg/l), (13women with hs-CRP 3 -6mg/l) and (20women with hs- CRP <3mg/l), also the same women were divided according to BMI into: (8 women with BMI>50), (9 women with BMI 41-49) and (33women with BMI 30-40). The results showed, that the TGs (pre-prandial and postprandial), and anthropometric measurement were significantly ($p \leq 0.05$) increased in (17 women with hs-CRP> 6mg/l), while in (13women with hs-CRP 3 -6mg/l) and (20women with hs- CRP <3mg/l) were decreased. Also the results revealed that, TGs, hs-CRP and anthropometric measurement were significantly ($p \leq 0.05$) increased in (8 women with BMI>50) as compared with (9 women with BMI 41-49) and (33women with BMI 30-40) respectively. It was concluded that, hs-CRP and (BMI), are a features of postmenopausal women obesity.

KEY WORDS: hs-CRP, (BMI), Obesity, WHR, Postmenopausal.

INTRODUCTION

Obesity represents one of the great modern health challenge defined as an excessive fat accumulation in adipose tissue, to the extent that health may be impaired. The underlying cause is the undesirable positive energy balance and weight gain. However, obese individuals differ in the amount of excess fat that they store, and in regional distribution of that fat within the body. The distribution of fat induced by weight gain affect the risks associated with obesity and kinds of disease that result [1, 2]
Obesity is a state of extreme fatness and is classified as a body mass index of greater than or equal to (30) with the highest class of obesity having a BMI equal or greater than (40) in adults [3]. The BMI is defined as measure of adult's weight (body mass) relative to height, used to assess the extent of weight deficit or excess where height and weight have been measured. BMI is the weight in kilograms divided by the square of the height in meters[4].Central type of obesity is linked to the shape of an apple: the shoulders, face, arms, neck, chest and upper portion of the abdomen are bloated. The stomach gives appearance, so also the arms, shoulders, and breasts. The lower portion of the body, hips, thighs, and legs are thinner in comparison with upper part. In these persons, the vital organs affected will be mostly the heart, liver, kidneys and lunges [5].Central obesity is also associated with increased production of cholesterol, primarily in the form of low-density lipoprotein, leading to increased incidence of atherosclerotic cardiovascular disease and gallstones[6]. Persons with peripheral type of obesity have extra flesh in their lower parts. This type is found in males and females, but more common among females, the flesh is flabby in the abdomen, thighs, buttocks, and legs. The face and neck mostly give a normal appearance. Peripheral type of obesity is a major risk for the uterus, intestine, kidneys, bladder, and bowls are the mostly affected, but the function of these organs sometimes has a direct effect on the heart[7]. A mixed central-peripheral phenotype is characterizes by an overall increase in fat accumulation, severely obese women and men (BMI>40 Kg/m2) may both present with this phenotype [8].
C-reactive protein (CRP) is the prototype a cute – phase protein primary synthesized in the liver and its release is stimulated by interleukin 6(1L-6) and other pro-inflammatory including increased activation of leucocytes and associated changes in inflammatory cytokine production[9].C-reactive protein has a normal concentration of less than 2mg \L in healthy -individuals. In the presence of infections or illness such as sepsis or rheumatoid arthritis the level of CRP increase sharply in the first 6-8 hours and can reach peak levels approaching 300mg\L after approximately 48 hours[10] plasma half-life of CRP is quite short at about 19 hours[11], plasma half-life and fractional clearance rates of CRP are nearly constant in normal subject, as well as in patients with infections, inflammatory and neoplastic conditions. It has been found that CRP concentration are actually relatively constant in an individual, both with regard to time of day, and over

days and months, even over months to years[10]. The clinical significance of CRP is not gender- specific. However; women appear to have higher plasma CRP levels than men do. Inflammation plays a role in the development of atherosclerosis and coronary heart disease [12]. As adipose is a major secretory organ for proinflammatory cytokines, obesity is consider to be a state of low-level inflammation. The relationship between obesity and inflammation is, in part, reflected by its close relationship with plasma concentration of CRP a sensitive marker for a cute –phase systemic inflammation. Studies show that elevated serum high – sensitivity (hs- CRP) concentrations are a significant predictor for incidence of the metabolic syndrome [13] , and type2 diabetes[14]. Hs-CRP has also been linked with coronary heart disease. Hs-CRP not only may be a marker of low grade chronic systemic inflammation but also may be directly involved in atherosclerosis. It can amplify the anti-inflammatory response through complement activation, tissue damage, and activation of endothelial cells[15].

MATERIALS & METHODS

Patients

Fifty female patients with obesity disease were randomly chosen from people attended the consulting clinic of Al-Kindy Teaching Hospital and Obesity Research and Treatment Unit of Al-Kindy College of Medicine / University of Baghdad. The diagnosed of patients as having obesity were based on the history, anthropometric measurements and clinical examination. Measurement of anthropometric variables in all participants was done according to standard methods[16].

Collection of blood samples

Blood samples were collected from each patient on the following basis: Fasting test (10-14 h before breakfast), two hours postprandial sample (with standardized breakfast) and four hours postprandial sample. In each stage, ten milliliters of venous blood were withdrawn from each obese patient .The samples were dropped into clean disposable tubes, left at room temperature for 15 minutes for clot formation and then centrifuged for 10 minutes at 3000 run per minute. The serum was separated and Triglyceride was measured by used (kit Randox England) and High sensitive C-reactive protein by (Linear Spain kit).

Statistical Analysis

The Statistical Analysis System-SAS (2010) was used to detect difference factors in study parameters (applied of completely randomized design-CRD). Duncan's test [17] was used to find the difference factors in study parameters.

RESULTS

High Sensitive C - reactive protein

In the present study table (1) showed that, the very high level of hs-CRP (8.19 ±0.19 mg/l) was found in 17 obese postmenopausal women (hs-CRP >6 group). The moderately increase level of hs-CRP (4.43±0.21 mg/l) was found in 13 obese postmenopausal women (hs-CRP 3-6 group), While, the mild levels of hs-CRP (1.63 ±0.13 mg/l) was found in 20 obese postmenopausal women (hs-CRP <3 group). From the same table a significant difference (P<0.05) was found between the level of TG (178.18±5.66 mg/dl) in (hs-CRP >6 group) and the levels of TG (115.38±5.29 mg/dl) in (hs-CRP 3-6) and (92.60±2.19 mg/dl) in (hs-CRP <3 groups). Moreover, the current study showed that the significant elevation of TG-2h (235.18±17.90 mg/dl) and TG-4h (337.74±38.35 mg/dl) in (hs-CRP >6 group) as compared with the levels of TG-2h (147.62±5.1 mg/dl) and TG-4h (198.46±6.97 mg/dl) in (hs-CRP 3-6) and TG-2h (120.10±4.97 mg/dl) and TG-4h (152.45±3.91 mg/dl) in (hs-CRP <3 groups) respectively. Also table (1) showed a significant difference (p<0.05) in the mean BMI of severe hs-CRP (50.02± 1.99 kg/m2) as compared to mild hs-CRP (32. 91 ± 0.33 kg/m2) and moderate hs-CRP (36.37±0.36 kg/m2), While there is no significant differences (p>0.05) for the same parameter between moderate and mild hs-CRP groups. A significant differences (p<0.05) between the WHR in severe hs-CRP group (1.26± 0.63), mild (0.86 ± 0.02) and moderate (0.97 ± 0.036) of hs- CRP groups. The results of WHtR showed that a significant difference (p<0.05), in the means of WHtR of mild hs-CRP (0.63± 0.02), moderate hs-CRP (0.72 ± 0.02) and severe hs-CRP groups (0.86 ± 0.35).A significant differences (p<0.05) among the mean of NC of mild hs-CRP (36.95 ± 0.17cm), moderate hs-CRP (39.0.7± 0.39) and severe hs-CRP (42.30± 0.32cm)

.

TABLE 1: Comparison of different parameters in Hs-CRP groups in obese postmenopausal women (Mean ± S.E.)

Groups →	(Hs-CRP < 3)	(Hs-CRP 3-6)	(Hs-CRP >6)
Parameters ↓	No=20	No=13	No=17
Age (year)	51.15 ± 1.20 (B,C)	55.38 ± 1.86 (A)	58.29 ± 1.14 (A)
TG (mg/dl)	92.60 ± 2.19 (B,C)	115.38 ± 5.29 (A,C)	178.18 ± 5.66 (A,B)
TG-2h (mg/dl)	120.10 ± 4.97 (C)	147.62 ± 5.1 (C)	235.18± 17.90 (A,B)
TG-4h (mg/dl)	152.45 ± 3.91 (C)	198.46± 6.97 (C)	337.74 ± 38.35 (A,B)
Hs-CRP (mg/l)	1.63 ± 0.13 (B,C)	4.43± 0.21 (A,C)	8.19 ± 0.19 (A,B)
BMI (kg/m2)	32.91 ± 0.33 (C)	36.37 ± 0.36 (C)	50.02 ± 1.99 (A,B)
WHR	0.86 ± 0.02 (C)	0.97 ± 0.036 (C)	1.26 ± 0.63 (A,B)
WHtR	0.63 ± 0.02 (B,C)	0.72 ± 0.02 (A,C)	0.86 ± 0.35 (A,B)
NC (cm)	36.95 ± 0.17 (B,C)	39.07 ± 0.39 (A,C)	42.30 ± 0.32 (A,B)

A: Significant mild group with other groups at level P < 0.05 / B: Significant moderate group with other groups at level P < 0.05 /C: Significant severe group with other groups at level P < 0.05.

Body Mass Index

The highest BMI (57.09±2.26 kg/m2) was found in 8 of obese (sever BMI group), the moderate BMI was observed in 9 obese (43.07± 0.78 kg/m2) (moderate BMI group) and the lowest to BMI was seen in 33 obese (34.27± 0.38 kg/m2), (mild BMI group). Table (2) showed that a significant difference of TG (mg/dl), TG-2h (mg/dl) and TG-4h (mg/dl) among all BMI groups (P< 0.05). The higher significant (P< 0.05) levels of TG (197.63±4.78 mg/dl), TG-2h (290± 27.01 mg/dl) and TG-4h (446.25 ±

62.11 mg/dl) was found in severe BMI group as compared with mild and moderate BMI groups. The lowest significant(P< 0.05) levels of TG (101.58±3.12 mg/dl), TG-2h (130.94 ±4.28 mg/dl) and TG-4h (170.58 ± 5.33 mg/dl) was found in mild BMI group as compared with the levels of TG, TG-2h, TG-4h in severe and moderate BMI groups(P<0.05). A significant differences (p<0.05) between the mean WHR of mild BMI, moderate BMI and severe BMI groups and the value of these group were (0.9 ± 0.02), (1.16± 0.06) and (1.37 ±0.11) respectively. A significant difference (p<0.05),in the WHtR among mild, moderate and severe BMI groups with value of (0.66 ± 0.2kg/m2),(0.77 ± 0.02 kg/m2) and (0.96 ± 0.05 kg/m2) respectively. Also a significant differences (p<0.05) was found in NC in mild, moderate and severe BMI groups, and the values were (37.79 ± 0.26 cm) ,(41.33 ± 0.24 cm) and (43.38 ± 0.32 cm) respectively.

TABLE 2: Comparison of parameters in BMI groups in obese postmenopausal women (Mean ± S.E.)

Groups →	Mild	Moderate	Severe
Parameters ↓	BMI (30-40) No=33	BMI (41-49) No=9	(BMI ≥ 50) No=8
Age (year)	52.82 ± 1.08 (B,C)	57.33 ± 1.27 (A)	59.38 ± 1.99 (A)
TG (mg/dl)	101.58 ± 3.12 (B,C)	160.88 ± 4.86(A,C)	197.63 ± 4.78 (A,B)
TG-2h (mg/dl)	130.94 ± 4.28 (B,C)	186.44 ± 3.30(A,C)	290 ± 27.01 (A,B)
TG-4h (mg/dl)	170.58 ± 5.33 (B,C)	241.33 ± 9.23(A,C)	446.25 ± 2.11(A,B)
Hs-CRP (mg/l)	2.73 ± 0.27 (B,C)	7.63 ± 0.15 (A,C)	8.81 ± 0.22 (A,B)
BMI (kg/m2)	34.27 ± 0.38 (B,C)	43.07 ± 0.78 (A,C)	57.09 ± 2.25 (A,B)
WHR	0.90 ± 0.02 (B,C)	1.16 ± 0.06 (A,C)	1.37 ± 0.11 (A,B)
WHtR	0.66 ± 0.2 (B,C)	0.77 ± 0.02 (A,C)	0.96 ± 0.05 (A,B)
NC (cm)	37.79 ± 0.26 (B,C)	41.33 ± 0.24 (A,C)	43.38 ± 0.32 (A,B)

A: Significant mild group with other groups at level P < 0.05 / B: Significant moderate group with other groups at level P < 0.05 / C: Significant severe group with other groups at level P < 0.05.

DISCUSSION

High Sensitive C - reactive protein

Obesity is determined by increased system concentration of inflammatory markers and cytokines in patients and animal models of obesity, the main of this systemic inflammatory response is shown to be located in adipose tissue[18]. Adipose tissue produces a number of inflammatory cytokines, and some of them are found elevated in the serum of obese patients. Elevated markers of inflammation are associated with an increased risk of future cardiovascular disease [19]. Triglyceride levels were greater in hs-CRP > 6 group as compared to TG levels in hs-CRP < 3 group and hs-CRP 3-6 group all times. This is consistent to the finding of [20, 21]. they suggested that unfavorable lipid profile may facilitate the formation of foam cells in the arterial wall, increasing the inflammatory activity and increased hs-CRP [22], observed a significant positive correlation between CRP and TG in Korean females aged 18-64. [23] indicated that increase in hs-CRP levels with dyslipidemia profile in the Indian population [24,25], concluded that a positive correlation existed between the level of hs-CRP and TGs concentration and a negative correlation existed between hs-CRP level and HDL-C concentration in the middle – aged group (40-59).

Present study showed that the maximum level of BMI was found in sever hs-CRP group .This finding in current study proved the direct relationship between level of BMI and level of hs- CRP. These results agree with previous studies as seen in study done by [26, 27] , they indicated that CRP is strongly associated with BMI. Also, [28] found that the hs-CRP level was significantly correlated with BMI level in females with and without diabetes[29], concluded that metabolic factors especially BMI had a relatively strong associated with hs-CRP at all ages. The lower level of WHR was seen in mild hs-CRP group followed by to moderate. While, the highest level was seen in severe hs-CRP groups, the current study showed that the increase in the WHR value was combined with increasing of hs-CRP level. These findings were agreed with other similar works [30], showed that the relation among of CRP level with WHR and other correlates of the metabolic syndrome [31] , reported a marginal association between WHR and CRP among elderly Taiwanese women aged 65 and oldest and [28] showed that the hs-CRP level was significantly correlated with waist circumference and WHR values among females with and without diabetes,

In the present study the greater level of WHtR was observed in severe hs-CRP group. These findings agree with previous results of [32], they concluded that WHtR and ultrasensitive CRP were predictors of the presence of the metabolic syndrome in children. In addition that [33], showed that WHtR values and other anthropometric measurement were in positive and linear associated with inflammatory markers such as CRP, TNF soluble, TNF-receptor 1, and IL-6 . In present study, NC is positively associated with raising hs-CRP groups. This was in accordance with[34], concluded that NC and other anthropometric measurement were identified as risk anthropometric measurement and identified as risk factor of hs-CRP in obstructive sleep Apnea syndrome and [35] revealed that appositive relationship between NC and metabolic syndrome with increasing hs-CRP. The present study showed that a degree of inflammation appeared difference in terms of intensity, which was divided into mild, moderate and severe.

Body Mass Index

Body Mass Index has traditionally been used to identify individuals who are the most likely to be overweight or obese, generally a high value BMI indicates excessive body fat and consistently relates to increased health risks and mortality. The results of current study showed positive relationship between the BMI and TG, TG-2h and TG-4h levels. These results agree with the results of [36], they noticed that increase in dyslipidemia was concomitant with increasing in BMI in both females and males. Other study reported an increase of postprandial triglyceridemia response with higher visceral obesity compared to others with lower visceral adiposity [37].

The current study showed that an increase in the WHR value was concomitant with increasing in BMI values. These findings agree with other similar works [38, 39], found that the body mass index of the menopausal women was positively correlated with the waist circumference and WHR since increased visceral and subcutaneous fat in menopause.Moreover fat is deposited around the abdomen more than at the hips as seen in women in the reproductive age which presents as greater increases in fat mass and WHR. In the present studies, the highest level of WHtR was found in severe BMI group, WHtR rose with increasing BMI value. These findings were in the line with previous study of [40], who pointed out that WC, WHtR and BMI values were associated with metabolic risk factors, and they may equally predict multiple metabolic risk factors, While [41], referred that BMI and WHtR were found to be the most significant obesity parameters for predicting insulin resistance in Jordanians male and female populations.

The highest level of NC was found in severe BMI group, NC is positively associated with BMI values. This is in accordance with previous studies [42], found that highly significant correlation between BMI and NC as well as between BMI and the abdominal circumference, which implies that the amount of body fat was related to the circumference [43], found that NC was positively related with BMI, WC, and metabolic syndrome in Chinese subjects having types2 diabetes mellitus[44], showed a strong positive correlation of NC with BMI and WC in both male and female subjects and NC was a potentially useful initial screening tool for overweight /obesity. Generally, by the current results greater BMI increases the side effects of a high level of fat and inflammation and the rest of the anthropometric parameters, for this there is a direct correlation was found between BMI and the parameters mentioned previously in this study.

CONCLUSION

From the above results it can be concluded that the body mass index and Hs-CRP are good predictors for obesity in postmenopausal women

REFERENCES

[1]. Wolin, K.Y. and Petrelli, J.M. (2009) Obesity. Greenwood group. USA; p. 28.

[2]. Rippe, J. and Angelopoulos, T. (2012) Obesity prevention and treatment. *CRP Press*, P. 1-30.

[3]. Bagchi, D. and Preuss. H. (2013) Obesity: Epidemiology, pathophysiology, and prevention.2nd ed. *CRP pree*; P. 1-10.

[4]. Hersen, M. (2011) Clinician's Handbook of Adult Behavioral Assessment. Gulf Professional Publishing, P.261.

[5]. Schnieder, M. (2010) Inroduction to public Health. Jone and Bartlett learning;, P. 264.

[6]. Arora, A. (2007) 5 steps to manage obesity: are you tired of being overweight.Sterling publishing *PVT. Ltd*; P.17-18.

[7]. Savard, M. (2007) Apples and Pears: The body shape solution for weight loss and wellne. *Simon and Schuster*, P. 1-35.

[8]. Iacobellis, G. (2009) Obesity and Cardiovascular Disease.Oxford University Press, P. 46.

[9]. Daniel, G.H. and Sonia S.A. (2003) Emerging risk factor atherosclerotic disease: A critical review of the evidence. *Journal of Amrican Heart Association*; **290**: 932-940.

[10]. Li, J.J. and Fang, C.H. (2004) C-reactive protein is not only an inflammatory marker but also a direct cause of cardiovascular disease. *Medical Hypothesis*; **62**: 499-506.

[11]. Lacsan, E. and Levint, N. (2004) C-reactive protein and end-stage renal disease. *Seminars in Dialysis* **17**: 438-448.

[12]. Lind, L. (2003) Circulating markers of inflammation and atherosclerosis,; **169**:203-214.

[13]. Han, T., Sattar, N., Williams, K., Gonzalez-Villapando, C., and Haffner, S. (2002) Prospective study of C-reactive protein in relation to the development of diabetes and metabolic syndrome in the Mexico City Diabetes Study. *Daibetes Care*.; **25**:2016-2012.

[14]. Hu, F., Meigs, J., Li, T., Rifai, N. and Manson, J. (2004) Inflammatory markers and risk of developing type 2 diabetes in women. *Diabetes*. **53**:693-700.

[15]. Libby, P., Ridker, P. and Maseri, A. (2002) Inflammation atherosclerosis. *Circulation*, **105**: 1135-1143.

[16]. Lohman, T., Roche, A. and Martorell, R. (eds.) (1998) Anthropometric Standardization Reference Manual. Human Kinetics Books: *Champaign, IL ISBN* O-87322-121-4.

[17]. Duncan, D. (1955) Multiple range and multiple F test. *Biometric*, 11:1-42.

[18]. Kiefer, F., Zeyda, M., Todroric, J., Huber. J., Geyergger, R., Weichhart, T., Aszmmann, O., Ludvik, B., Silberhumer, G.R., Prager, G. and Stulnig. T.M. (2008) Osteopontin expression in human and murine obesity: Extensive local up-regulation in adipose tissue but minimal systemic alterations. *Endocrinology*; **149** (3):1350- 1357.

[19]. Thakur, S. (2011) Hs-CRP - A Potential Marker for Coronary Heart Disease. *Indian Journal of Fundamental and Applied Life Sciences*, 1 (1); 1-4.

[20]. Horne, B., Muhlestein, J., Carlquist, J. (2000) Statin therapy, lipid levels, C-reactive protein and the survival of patients with angiographically severe coronary artery disease. *J Am Coll Cardiol.*; **36**:1774-80.

[21]. Francisco, G., Hernandez, C., Chacon, P., Mesa, J., and Simo, R. (2005) Factors influencing CRP levels in the diabetic population. *Med Clin (Barc*); **124** (9):336-7.

[22]. Choi, E.Y., Park. E.H., Cheong, Y.S. (2006) Association of C-reactive protein with the metabolic risk factors among young and middle – aged Koreans. *Metabolism*; **55**(3), 415-21.

[23]. Jeemon, P., Prabhakaran, D., Ramakrishnan, L., Gupta, R., Ahmed, F., Thankappan, K. (2011) Association of high sensitive C-reactive protein (hsCRP) with established cardiovascular risk factors in the Indian population. *Nutr Metab (Lond)* 8:19.

[24]. Haddad, N.S. (2012) High sensitivity C-reactive protein (Hs-CRP) and metabolic syndrome: correlation with number and type of metabolic syndrome components in Iraqi patients. *MJBU*; **30**(1).

[25]. Yi, Z., Ming, S.X., Michael, F.S., Zhu, Q.H., Virginia, B.K., Melanie, S., Perry, H., Wei, X.J. and Yi, Z. (2009) High Sensitivity C-reactive Protein Associated with different health predctors in Middle –Aged and oldest Chinese. *Biomed Environ Sci*; **25**(3):257-266.

[26]. AL-Kirwi, E. and Abed, B. (2009) Hypertension and obesity in relation to high sensitivity C-reactive protein and Lipid profile in Iraqi patients. *Journal of Al-Nahrain University*; **12**(4):145-150.

[27]. Hussein, W., Al-Bayatti, A. and Salman, E. (2009) High sensitivity C-reactive protein is a Significant Predictor for Hypertension and Obesity in Iraqi Postmenopausal Women. *J Fac Med Baghdad*; **5**1(3).328-331

[28]. Huffman, F., Whisner, S., Zarini, G., Nath, S. (2010) Waist Circumference and BMI in Relation to Serum High Sensitivity C - reactive protein (hs-CRP) in Cuban Americans with and Without Type 2 Diabetes. *Int J Environ Res Public Health*; **7**(3):842-52.

[29]. Laugsand, L., Asvoid, B., Vatten, L., Romundstad, P., Wiseth, R., Hveen, k., and Lansky, I. (2012) Metabolic factors and high -sensitivity Creactive protein: the HUNT study. *European Journal of preventive Cardiology*; **19**(5):1101-1110.

[30]. Patel, D.A., Srinivasan, S.R. Xu, J. H. Li S. and Berenson, G.S. (2006) Distribution and metabolic syndrome correlate of plasma C-reactive protein in biracial (black-white) younger adults: the Bogalusa Heart Study. *Metab. Clin. Experim.* **55**, 699-705.

[31]. Tsai, H.J., Tsai, A.C. and Tasai, A.C. (2008) The association of plasma Creactive protein levels with anthropometric and lipid parameters in elderly Taiwanese. *AsiaPac J Clin Nutr,;* **17**(4), 651-6.

[32]. Arnaiz, P., Marína, A., Pinoa, F., Barja, S., Aglony, M., Navarrete, C., and Acevedo, M. (2010) Waist height ratio, ultrasensitive c reactive protein and metabolic syndrome in children. *Rev Med Chile*;, **138**: 1378-1385.

[33]. Wu, S., Shu, X.O., Chow, W., Xiang, T., Zhang, X., Cai, Q., Li, H., Milne, G., Wen, W., Ji, B., Gao, Y., Rothman, N., Zheng, W. and Yang, G. (2013) Adiposity and fat distribution in relation to inflammatory and oxidative in a relatively lean population of Chinese women. *Dis Markers***, 34**(4): 279-93.

[34]. Chung, S., Yoon, I., Shin, Y, Lee, C.H., Kim, J, Lee, T., Choi D, and Ahn, H.J. (2007) Endothelial Dysfunction and C-Reactive Protein in Relation with the Severity of Obstructive Sleep Apnea Syndrome .*National Institutes of Health*;, **30**(8):997-1001.

[35]. Vallianous, N., Evangelopolos, A., Bountziouka, V., Vogiatzakis, E., Bonou, M., Barbetseas, J., Avgerinos, P. and Panagiotakos, D. (2012).Neck circumference is correlated with triglycerides and inversely related with HDL- cholesterol beyond BMI and waist circumference. *Diabetes Metabolism Research*;, **29** (1):90– 97.

[36]. Brown, C.D., Higginsb, M., Donato, K.A., Rohde, F.C., Garrison, R., Obarzanek, E. (2000).Body Mass Index and the Prevalence of Hypertension and Dyslipidemia *Obes Res*., **8**(9):650-19.

[37]. Lairon, D.L. (2009) Metabolism postprandial des lipides: une autre vision sur les relations alimentation métabolismesanté. *CERIN*, 112:1-6.

[38]. Achie, L.N., Olorunshola, V.K., Toryila. E.J. and Tende, J.A. (2012) The body mass index, waist circumference and blood pressure of postmenopausal women in Zaria, Northern Nigeria. *Curr. Res. J. Biol. Sci*., **4**(3): 329-332.

[39]. Gandhi, R., Dhotar, H., Tsvetkov, D. and Mahomed, N.N. (2010) The relation between body mass index and waist-hip ratio in knee osteoarthritis. Can J Surg;, **53** (3): 151-4.

[40]. Liu, Y., Tong, G., Tong, W., Lu, L. and Qin, X. (2011)Can body mass index, waist circumference, waist –hip ratio and waist –height ratio predict the presence of multiple metabolic risk factor in Chinese subjects. *BMC Public Health*; ,11:35

[41]. Abu khadra, K. and Aljaberi. A. (2012) nthropometric measures as predictors for the occurrence of insulin resistance among obese Jordanians. *Scientific Research and Essays;*, **7**(25): 2218- 2224.

[42]. Mueller, W.H., Wear, M.L., Emerson, J.B., Barton, S.A., Hewett- Emmett, D. and Schull, W.J. (1991) Which measurement of body fat distribution is best for epidemiologic research. *AmJ Epidemiol*, **133**:858-869.

[43]. Yang, W., Lu, J., Weng, J., Jia, W., Ji, L., Xiao, J. (2010) Prevalence of Diabetes among Men and Women in China. *N Engl J Med*; **362**:1090-101.

[44]. Hingorjo, M.R., Qureshi, M.A. and Mehdi, A. (2012). Neck circumference as a marker of obesity: A comparison with body mass index and waist circumference. *J Pak Med Assoc;*, **62**(1):36-40.